OLIGURIA AND LEFT UPPER LIMB WEAKNESS

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A 32 year old male had his first experience with heroin whilst under the influence of alcohol. He fell into a deep alcoholic slumber and lay motionless on his left side with his left arm tucked under his body, remaining thus till the morning.

He regained consciousness in hospital where he became aware of his swollen, motionless left upper limb which felt numb. He had severe vomiting and also noted that his urine output had decreased. His urine was found to be reddish having proteinuria (300 mg%), occult blood positive and the spun sediment showed a few RBC's with many granular and pigmented casts.

Conservative measures having failed to prevent anuria, he was referred for dialysis. Physical examination revealed a normal temperature, a pulse rate 88/min. and a blood pressure 140/90 mm Hg. His swollen left upper limb had lost movement at all joints. The muscles of the upper arm were slightly tender on palpation. The limb had a pitting edema. All sensory modalities had been lost over the forearm, hand and over most of the upper arm except for a small area over the left deltoid. Rest of the clinical examination (including pulses in the affected limb) was normal.

Laboratory investigations showed a white blood cell count of 9300/cmm. of which neutrophils were 77% Hematocrit and haemoglobin values were 51% and 16.8 gm% respectively.

Biochemical examinations revealed urea nitrogen 61%, creatinine 9.5%, total protein 6.2 gm%, albumin 3.4 gm%, normal bilirubin levels, sodium-138 mEq/L, potassium 5.8 meq/L, chloride 86 meq/L, plasma bicarbonate 21 m.mol/litre, uric acid 9.8 mg%, calcium 8 mg%, phosphorous 8 mg%, alkaline phosphatase 4.0 bodansky units.

Enzymes studies showed serum transaminases (SGOT, SGPT) greater than 250 units (normal 15-40 units), LDH 1035 mm/ml. (0-300 mm/ml.) and CPK (24.99 Um/ml./hr. (0.4—4.0 Um/ml./hr.).

Ultrasound examination of the kidneys revealed normal sized kidneys with normal pelvicalyceal system.

An EMG showed spontaneous fibrillation potentials (neurogenic injury) and low voltage action potentials (myogenic injury) in the left upper limb.

The patient was placed on hemodialysis as long as he remained oligoanuric. His left upper limb remained swollen and hence, to prevent vascular compression, fasciotomy incisions were done in the forearm and arm.

About the 15th day after renal shutdown, he went into the diuretic phase. Investigations done 2 days after entering the diuretic phase revealed hemoglobin 7.2 gm%, total leucocyte count 8900/cmm. with neutrophils 69%. Urinalysis revealed traces of albumin, 1-2 pus cells/HPF, BUN 88 mg%, creatinine 9.5 mg%, total proteins of 5.6 gm%, albumin 3 gm%, normal bilirubin and electrolyte levels. Uric acid 6.5 mg%, calcium values 15.0 mg% and phosphorous 6.5 mg%. SGOT and SGPT levels were 50 units and CPK levels had fallen to 6.6 Um/ml/hr.

The patient thereafter, had a rapid return of renal function. The paralysed left upper limb did not show as rapid a recovery. Minimal movements had returned at the wrist, elbow and shoulder joint with no movements at the fingers. With neurological recovery, the patient had severe pain which required large doses of analgesics and also epidural morphine injections. He was receiving the physiotherapy till the time of discharge from the hospital.

DISCUSSION

This 32 years old previously healthy, well-built and nourished male, presented with acute renal shutdown and paralysis of the left upper limb.

Q.1. What are the possible factors that could have produced acute renal failure in this patient?

He could have suffered acute renal injury following volume depletion or through the action of alcohol and heroin alone or in combination.

From the available data, there is a history of incessant vomiting following 'drinks' and poor intake of food. Thus, it may be surmised that a certain degree of volume depletion did exist during the insult or induction phase of acute renal failure. At no stage was the hypovolemia severe enough to induce a state of hypovolemic shock. It therefore, implies that probably there existed other factors that accentuated or supplemented the renal injury.

The history and clinical findings along with the laboratory investigations aid us in coming to the

conclusion that alcohol and/or heroin intoxication had contributed maximally to the induction of renal injury. When the patient had developed oliguria his urine was reddish with proteinuria (300 mg%) and showed occult blood positive test. There was no significant microscopic hematuria along with pigmented casts. Biochemical tests done revealed disproportionate elevation of serum creatinine (normal ratio of BUN to creatinine being 10 : 1) with respect to BUN, hyperkalemia, hyperuricemia, hypocalcemia, hyperphosphatemia, elevated serum transaminases; LDH and CPK levels. A myogenic pattern was obtained in EMG studies of the left upper limb. These pointers indicate evidence of muscle injury (rhabdomyolysis) with release of myoglobin into circulation.

Q.2. Can rhabdomyolysis be recognised clinically?

Rhabdomyolysis (non-traumatic) is often a subtle process, unassociated with overt symptoms or physical findings. It may be associated with muscle pain, stiffness, swelling and weakness. When severe, deep tendon reflexes are absent.

For long, non-traumatic causes of rhabdomyolysis remained unrecognised and its only of recent, that they have been incriminated in the causation of myoglobinuric renal failure.

Q.3. What clinical situation should lead to the suspicion of myoglobinuric renal failure?

A patient in acute renal failure who had passed scanty, red coloured benzidine positive urine should have a serum sample examined to rule out hemoglobinuria. In the presence of a clear serum, hemoglobinuria is ruled out as a cause for the renal shutdown and incriminates myoglobinuria as the cause.

Q.4. Which newer methods have made a detection of myoglobinuria or myoglobin release into circulation possible?

Immunoprecipitation methods measure levels upto 0.005 mg% in urine and blood and these sophisticated methods have aided detection of rhabdomyolytic processes.

Q.5. Which patients are more susceptible to myoglobinuric renal failure?

Myoglobin contents of muscle varies with physical activity. Therefore, a healthy physically active person with rhabdomyolysis will have a more pronounced myoglobinuria and is much more likely to have acute renal failure than a poorly conditioned person.

Q.6. How does myoglobinuria cause renal failure?

The proposed pathogenetic mechanisms of acute renal failure in myoglobinuria are :

i) Tubular obstruction (myoglobin precipitates in concentrated acidic urine).

ii) Decreased renal blood flow and glomerular filtration rate (ischaemia); and

iii) Direct toxic injury to tubular epithelium.

Q.7. What were the causes for renal shutdown in this patient?

This patient had mild volume depletion with myoglobinuria following rhabdomyolysis resulting from alcohol intoxication and heroin administration under probably unsterile conditions.

Q.8. What management problems may be encountered in acute rhabdomyolysis?

The management problems could include fluid balances, renal insufficiency, hyperkalemia, hypocalcemia, hyperphosphatemia and disemminated intra vascular coagulation (DIC).

Q.9. What is the cause for volume depletion in rhabdomyolysis and what management is required?

Hypovolemia and shock results from loss of fluid into injured tissues. Replacement of losses with saline so as to maintain circulation and adequate urine output is all that is required.

Q.10. What measures should be offered to the myoglobinuric patient so as to preserve or prevent deterioration of renal function?

In the oliguric patient, it is important to look for prerenal cause (Urine Na $<$ 20 mEq/L; — suggests pre renal cause). In the presence of pre renal cause, fluids must be replaced. If even after fluid replacement, patient has developed tubular damage (urine Na $>$ 25 mEq/L), mannitol and frusemide must be used to prevent oliguric renal failure. Frusemide should be given in dose of 40-200 I.V. in conjunction with Mannitol (100 ml. of 25% solution) I. V. over 15 minutes. If no diuresis occurs Frusemide may be repeated in a dose of 200 mg. over 2 hours.

Q.11. What is the role of alkalis in rhabdomyolysis state?

Urinary alkalinisation to prevent myoglobin precipitation is not achievable in the absence of a metabolic alkalosis state. This in turn could result in more extensive precipitation of calcium salts in damaged tissues.

Q.12. Do electrolyte changes pose problems in acute rhabdomyolysis?

In severe rhabdomyolysis, hyperkalemia and acidosis co-exist. Ion exchange resins (orally-Kayexelate or aluminium sadolit 20 gms. three times a day or rectally retention enema of 60 gms. in 200 ml. of 10% glucose or sorbitol), glucose-insulin

infusions are the methods adopted to lower serum K+ levels. Dialysis may be resorted to in severe hyperkalemic states. Correction of acidosis is also important.

Hypocalcemia is prominent after 24 hours in severe rhabdomyolysis. The early hypocalcemia usually corrects itself spontaneously. Calcium infusions may lead to increased calcium salts precipitation in the injured tissues, and may fail to elevate serum calcium levels. Hyperphosphatemia is not severe and oral phosphate binders adequately control this state.

Q.13. Is a DIC state noted in acute rhabdomyolysis? What therapy is required?

A DIC state may be seen in rhabdomylosis. The natural course is of spontaneous recovery which usually begins on the third or fourth day. No significant benefit is obtained from heparin therapy.

Q.14. Does rhabdomyolysis involving the extremities create any special problems?

Careful observation for increasing edema and vascular compression is needed so that early fascistomy may be performed before extensive tissue necrosis occurs. Overlying rigidity and tenderness might well indicate impending necrosis. The behaviour of CPK activity (peaks on 2nd or 3rd day and thereafter, declined by half every 48 hours) could indicate the ongoing tissue necrosis.

Q.15. What causes hypercalcemia in the diuretic phase of myoglobinuric renal failure?

In the acute renal failure following rhabdomyolysis hypercalcemia is known to occure. Their hypercalcemic state is usually encountered in patients who have received calcium therapy for hypocalcemia during the few days post-muscle injury. The calcium that has been deposited in the injured muscle is remobilised and elevates the serum calcium levels. Hence, restricting the use of ion-exchange resins in calcium phase for correction of hyperkalemia, correction of dehydration together with frusemide administration helps in keeping hypercalcemia in control and the patient asymptomatic.

This patient required such therapy for hypercalcemia. Most of the medical problems listed were encountered in this patient and successful management was along the above mentioned lines.

DIET IN HEALTH AND DISEASE : by K. C. Patel and M. M. Prabhu, published by the authors, pages 66, price Rs. 15/-.

This is a small book which is easy to carry to wards. It deals with principles of diet in normal health and in some common medical and pediatric disorders. A sample diet sheet follows this. It is indeed creditable that the authors have covered this vast subject in a span of just 66 pages. The highlight of the book is that the diet sheet mentions common food stuff eaten by Indians etc., idli, jowar bhakra, khichedi etc.

The only problem with this book is that the style is rather jerky. Again the chapter on diet in normal health should be in a little more details. Printing errors have crept in and above all, the individual chapters have not been properly separated. The future editions should eliminate these problems.

For their maidan venture, the authors deserve compliments. The book is recommended to all medical, and nursing students and well as busy practitioners for a quick reference.

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